Original Contributions

Ultrasonographic Follow-up of Subclavian Stenosis and Occlusion: Natural History and Surgical Treatment

H. Ackermann, MD, H.C. Diener, MD, H. Seboldt, MD, and C. Huth, MD

Continuous-wave Doppler ultrasonography and clinical examination were used over a 2-year period to monitor the natural history of subclavian stenoses and occlusions in 67 patients. Thirty-nine presented with subclavian steal and 28 without. We also studied an additional group of 29 patients who had undergone surgery for subclavian steal syndrome and vertebrobasilar transient ischemic attacks. The results, in terms of both ultrasonographic and clinical criteria, demonstrate the benign nature of the subclavian steal syndrome: all neurologic signs and symptoms were of a transient character. Spontaneous remission of vertebrobasilar transient ischemic attacks occurred in approximately 50% of the initially symptomatic patients, and only 15% of the initially asymptomatic patients experienced vertebrobasilar transient ischemic attacks during follow-up. Doppler ultrasonography revealed progression during follow-up in only 17% of the subclavian stenoses, and in 13% a stenosis was no longer detectable. Still-ongoing brainstem transient ischemic attacks were reported in 24% of the operated patients. The most important factor for the lack of improvement was the occlusion of the bypass. Continuation of transient neurologic symptoms could be observed in only 14% of the patients with intact carotid-subclavian bypass. In conclusion, indications for surgical treatment of the subclavian steal syndrome should be restricted to cases in whom vertebrobasilar transient ischemic attacks occur frequently and are either debilitating or greatly frighten the patient. (Stroke 1988; 19:431-435)

Continuous-wave (CW) Doppler ultrasonography has proven to be a reliable method for the detection of subclavian stenoses or occlusions and for the documentation of retrograde blood flow in the vertebral artery (subclavian steal syndrome) or other collateral pathways to the subclavian artery.1-10 Due to inherent risks, angiography must be restricted to patients in whom surgery is considered or in whom recurrent stenosis or occlusion of a carotid-subclavian bypass is suspected. In contrast to angiography, the totally atraumatic ultrasonographic technique allows safe and routine follow-up of asymptomatic and symptomatic patients with subclavian stenosis or occlusion and of patients who have undergone surgery.

Several extrathoracic surgical approaches besides the older transthoracic procedures and the more recently outlined method of percutaneous transluminal angioplasty are recommended. Principally, surgery should provide relief from vertebrobasilar symptoms. There is, however, still considerable disagreement concerning the indication for surgical intervention. The most widely accepted assumption is that subclavian stenosis or occlusion with concomitant subclavian steal should be treated surgically when transient ischemic attacks (TIAs) attributable to the vertebrobasilar vascular region occur.11,12 But surgery is considered even in asymptomatic patients, provided that steal is demonstrated, arguing that normal conditions in the vertebrobasilar circulation may prevent symptomatic ischemia in the vascular region of the carotid arteries when carotid stenosis develops.13 However, other authors restrict surgery to patients with disabling vertebrobasilar TIAs.14

There is, however, up to now no scientific basis for the recommendation of bypass surgery in the presence of vertebrobasilar TIAs. Data about the natural history of patients with subclavian stenosis or occlusion may provide information concerning the priority of surgical intervention. As far as we know, there are only three short reports documenting the time course of subclavian steal syndrome.15-17 The aim of our study was to monitor ultrasonographic and clinical follow-up findings in patients with subclavian stenosis or occlusion over a period of at least 2 years. Our results were compared with those from a group of patients who underwent common carotid-to-subclavian artery bypass surgery.

Subjects and Methods

Between 1978 and 1985, 23,500 patient examinations involving CW Doppler ultrasonography were performed in the Department of Neurology at the

From the Departments of Neurology (H.A., H.C.D.) and Cardio-Vascular Surgery (H.S., C.H.), University of Tübingen, Tübingen, Federal Republic of Germany.

Address for correspondence: Hermann Ackermann, MD, Department of Neurology, University of Tübingen, Liebermeisterstraße 18-20, 7400 Tübingen, Federal Republic of Germany.

Received April 1, 1987; accepted December 1, 1987.
University of Tübingen. In 272 of these patients, a total of 308 subclavian stenoses or occlusions were detected. The ultrasonographic and clinical findings of this population have been reported. The term vertebrobasilar TIA was used in a broad sense, including also vertigo and short episodes of unconsciousness after exclusion of peripheral-vestibular and cardiac reasons (clinical examination, electrocardiography [ECG], calorics). Ultrasonographic and clinical follow-up data were available from 96 patients who were divided into four groups according to symptoms and treatment (Table 1).

**Group 1.** Twenty-nine patients (24 with subclavian occlusion and five with stenosis) underwent surgery for the relief of TIAs. A bypass from the adjacent common carotid to the subclavian artery was performed using a Dacron prosthesis. All Group 1 patients suffered from vertebrobasilar TIAs before surgery, and in all of them CW Doppler ultrasonography and angiography revealed a subclavian steal syndrome. Ultrasonographic and neurologic follow-up examinations were performed between 6 weeks and 25 months after surgery (mean 11 months).

**Group 2.** Thirteen patients with vertebrobasilar TIAs in whom CW Doppler ultrasonography revealed subclavian stenosis or occlusion with concomitant retrograde blood flow in the ipsilateral vertebral artery refused surgery. Five patients showed occlusion (bilateral in none), and the other eight showed stenosis (bilateral in six). Ultrasonographic and clinical data of at least two examinations over at least 2 (range 2-5) years were available in all Group 2 patients.

**Group 3.** Twenty-six patients with subclavian stenosis or occlusion and retrograde blood flow in the ipsilateral vertebral artery never experienced clinical symptoms and signs of vertebrobasilar insufficiency before entry into this study. Seven showed stenosis (bilateral in three), and 19 showed occlusion (bilateral in one); 11 showed concomitant carotid lesions. Follow-up data were available as in Group 2 (range 2-6 years).

**Group 4.** Twenty-eight patients had subclavian stenosis or occlusion without subclavian steal. Occlusion was detected in four and stenosis in 24. One patient had bilateral occlusion and two had bilateral stenosis. Follow-up data were available as in Groups 2 and 3 (range 2-7 years). As in the other groups, in all Group 4 patients subclavian stenosis or occlusion was localized proximally to the vertebral artery origin.

Only >50% stenoses and occlusions of the internal and common carotid arteries were taken into account. Lesions of the external carotid arteries have no hemodynamic impact on vertebrobasilar insufficiency. For Groups 1-3 the mean difference in blood pressure was approximately 40 mm Hg (37 mm Hg for Group 1, 43 mm Hg for Group 2, 41 mm Hg for Group 3). In Group 4 the mean difference in blood pressure was only 20 mm Hg, reflecting the higher incidence of slight stenosis in this group.

A directional CW Doppler device (Debitmètre ultrasonique Delalande) with a frequency of 4 MHz was used for ultrasonographic examination. Blood flow directions and velocities were measured bilaterally in the supratrochlear, external, internal, and common carotid, subclavian, and vertebral arteries. The subclavian artery was investigated in the supraclavicular fossa. By tilting the Doppler probe, it is possible to obtain signals from the proximal and distal portions of the artery as well. The vertebral artery was insonated at the level of the atlas loop. Doppler signals were acoustically evaluated and the blood flow velocity was recorded on a strip chart. The direction of blood flow in the vertebral artery was determined by a functional test. Deceleration or acceleration of flow in the vertebral artery after inflation and release of a pressure cuff around the ipsilateral upper arm indicates reversed blood flow. We did not distinguish between permanent and intermediate stages of subclavian steal. A >50% stenosis of the proximal subclavian artery was assumed when increased flow velocity and poststenotic turbulence were audible. Severe stenosis results in reduced poststenotic systolic blood flow and decreased diastolic backflow. Occlusion was diagnosed when the combination of a >50% decrease in systolic flow, a loss of diastolic backflow, and a centrifugal flow during the mid- and end-diastole were observed.

Angiography was performed in 38 (all 29 in Group 1 and 9 of 13 in Group 2) of the 96 patients. In 35 of the 38 contrast medium was injected via a catheter placed at the origin of the subclavian artery (transfemoral access). Intravenous digital subtraction angiography was carried out in the remaining three patients.

**Results**

Table 2 compares the findings from ultrasonographic and angiographic examinations of 38 patients. CW Doppler ultrasonography did not result in false-

---

**Table 1. Ultrasonographic and Clinical Data for 96 Patients Examined With Continuous-Wave Doppler Ultrasonography**

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>n</th>
<th>Mean age</th>
<th>Range</th>
<th>N</th>
<th>Mean age</th>
<th>Range</th>
<th>Occlusion</th>
<th>Stenosis</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>29</td>
<td>22</td>
<td>55.7</td>
<td>36-69</td>
<td>7</td>
<td>55.5</td>
<td>45-67</td>
<td>24 (2)</td>
<td>5 (0)</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>13</td>
<td>8</td>
<td>58.3</td>
<td>40-78</td>
<td>5</td>
<td>59.6</td>
<td>44-65</td>
<td>5 (0)</td>
<td>8 (3)</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>26</td>
<td>19</td>
<td>58.6</td>
<td>38-73</td>
<td>7</td>
<td>54.7</td>
<td>27-73</td>
<td>19 (1)</td>
<td>7 (3)</td>
<td>7</td>
</tr>
<tr>
<td>4</td>
<td>28</td>
<td>20</td>
<td>57.2</td>
<td>40-81</td>
<td>8</td>
<td>58.0</td>
<td>40-75</td>
<td>4 (1)</td>
<td>24 (2)</td>
<td>8</td>
</tr>
</tbody>
</table>

Group 1, with subclavian steal, with transient ischemic attacks (TIAs), with surgery; Group 2, with steal, with TIAs, without surgery; Group 3, with steal, without TIAs, without surgery; Group 4, without steal, without TIAs, without surgery. Numbers in parentheses are patients with bilateral subclavian stenosis or occlusion.
positive findings in the diagnosis of subclavian lesions (>50% stenosis or occlusion), but in 23% (7 of 30) of the ultrasonographically diagnosed subclavian occlusions angiography revealed severe stenosis. Subclavian arteries evaluated as normal by Doppler ultrasonography were never found to have a >50% stenosis as demonstrated by angiography. There was 100% agreement between the two procedures for detection of retrograde vertebral blood flow.

Table 3 summarizes the ultrasonographic findings in the 29 Group 1 patients. Doppler ultrasonography performed 1.5–25 (mean 11) months after common carotid-to-subclavian artery bypass surgery detected occlusion in 24% (7 of 29) and stenosis in 7% (2 of 29) of the patients. Despite successful surgical treatment (intact bypass including stenotic bypasses, no complaints), delayed or no restitution of orthograde flow in the vertebral artery was observed in three cases.

Clinical improvement was observed in 76% (22 of 29, Table 4) of the Group 1 patients. Four of the seven patients (57%) with persisting vertebrobasilar TIAs had an occluded bypass demonstrated by ultrasonography. In contrast, 57% (4 of 7) of the patients with occluded bypass complained of persisting TIAs, but only 16% (3 of 19) of those with intact bypass and orthograde vertebral blood flow reported ongoing TIAs.

Table 4 also shows ultrasonographic data from Groups 2, 3, and 4 monitored over a period of at least 2 years. Taken together, the nonsurgical groups presented with 47 stenoses (bilateral in eight) and 30 occlusions (bilateral in two). The progression to occlusion occurred in 17% (8 of 47) of the confirmed cases of subclavian stenosis. In 13% (6 of 47) of the cases diagnosed at entry, stenosis was no longer detectable at a second examination. A total of 57 patients presented with unilateral stenosis or occlusion at the first investigation; during follow-up additional contralateral stenosis developed in 5% (3 of 57). In 7% (2 of 28) of the Group 4 patients retrograde vertebral blood flow was detected during follow-up.

No persisting neurologic signs or symptoms indicative of vertebrobasilar infarction were observed intraoperatively or perioperatively in Group 1 or during a ≥2-year follow-up in Groups 2, 3, and 4. In all four groups, neurologic signs and symptoms were of a transient character when present.

In Group 2 patients spontaneous remittance of TIA during follow-up occurred in 46% (6 of 13). In contrast, after follow-up of at least 2 years 15% (4 of 26) of the Group 3 patients reported newly manifested TIAs. Two Group 4 patients developed subclavian steal during follow-up. None suffered from vertebrobasilar TIAs.

**Discussion**

In interpreting the clinical results it must be considered that vertigo and blurred vision, the two
most-often reported vertebrobasilar symptoms in our patients, are not absolutely valid parameters indicating brainstem ischemia even if clinical examination, ECG, and calorics give no hint of cardiac or peripheral-vestibular causes. Further restrictions are consequent upon the retrospective design of our study since clinical symptoms were reported sometimes weeks to months after occurrence. Despite these obstacles to interpretation, there are two major conclusions resulting from our data. First and most important, the vertebrobasilar signs or symptoms did not persist in any patient.\textsuperscript{19} All signs and symptoms of vertebrobasilar insufficiency (short episodes of dizziness or blurred vision) observed or reported during follow-up were of a transient character and scarcely impaired daily life. Second, spontaneous remission markedly prevailed over occurrence of new vertebrobasilar TIAIs in our patients. Of the 13 Group 2 patients, 46% did not experience further problems attributable to vertebrobasilar insufficiency during follow-up. In addition, only 15% of the Group 3 patients reported first-time occurrence of vertebrobasilar TIAIs during follow-up. Thus, in roughly 50% of the patients with subclavian steal syndrome and vertebrobasilar TIAIs who met the usual criteria for surgical treatment, surgery would have been unnecessary. These data are in accord with the findings of Bornstein and Norris,\textsuperscript{17} who performed a prospective follow-up study in 45 asymptomatic persons with either the subclavian steal syndrome or "pre-steal." Only 11% of the 45 patients of Bornstein and Norris developed transient symptoms within a mean interval of 24 months, and none suffered a stroke.

Held et al\textsuperscript{15} noted a markedly different progression of the clinical symptoms of subclavian steal syndrome in men and women. They attributed this discrepancy to clinical data indicating more women with inflammatory vascular disease. In contrast, no difference in natural history between men and women was observed in our patients. This probably is due to the fact that in our patients, atherosclerosis was the predominant etiology of subclavian stenosis or occlusion in both men and women. In only one woman was Takayasu's syndrome diagnosed.

Ultrasonographic follow-up revealed a rather small tendency for the progression of subclavian stenosis. Only 17% of the subclavian stenoses developed to an occlusion, but 13% of the initially diagnosed stenoses were no longer detectable at follow-up examinations.

Among vascular surgeons there is some agreement that surgery should be restricted to patients with vertebrobasilar TIAIs.\textsuperscript{11,12,14} Our results from 29 patients with common carotid-to-subclavian artery bypass show that surgical treatment did not result in improvement of all cases: about one fourth of the operated patients (7 of 29) still suffered from the same complaints as before surgery. Intraoperatively and postoperatively neither death nor cerebrovascular accidents occurred.

Comparable clinical data of the outcome after surgery are reported in the literature.\textsuperscript{20-22} Other studies, however, alleged that continuation of vertebrobasilar TIAIs after surgery is very rare.\textsuperscript{23-31} In our group of surgically treated patients the mean age was rather high and the persisting vertebrobasilar symptoms usually involved vertigo. Even in patients with demonstrated subclavian steal syndrome and normal cardiology and otologic performance, it is nevertheless conceivable that intermittent vertigo not associated with retrograde vertebral blood flow occurs with advancing age. Discrepancies in terms of clinical improvement between groups of patients could be in part due to differences in the etiology of symptoms.

Previous reports have related recurrence of complaints after surgery to graft failure.\textsuperscript{29,31} Our data support this hypothesis. Posturgical ultrasonography detected an occlusion in 24% (7 of 29) of the grafts. Approximately 60% of our patients with occluded bypass but only 16% of those with intact bypass and orthograde ipsilateral vertebral blood flow complained of persisting postsurgical vertebrobasilar TIAIs.

It has been suggested that most patients with subclavian steal also have coexisting carotid artery disease.\textsuperscript{25,32,33} Some studies recommend reconstruction of ipsilateral carotid stenosis prior to common carotid-to-subclavian artery bypass or a one-stage surgical correction to prevent a siphoning effect of the bypass.\textsuperscript{34-36,37} In only four of our surgically treated patients, however, was concomitant carotid lesion detected by means of CW Doppler ultrasonography and angiography (contralateral internal carotid stenosis in two, occlusion of the ipsilateral internal carotid artery in the other two). A siphoning effect in terms of clinical events was not observed in any case.

The comparison of ultrasonographic and angiographic findings demonstrates the reliability of CW Doppler ultrasonography for the detection of subclavian stenosis and occlusion. No false-positive ultrasonographic diagnoses occurred in terms of >50% subclavian lesions at angiography; false-negative diagnosis on the contralateral side in patients with unilateral subclavian stenosis or occlusion was also not observed. Discrepancies between Doppler ultrasonography and angiography did occur, however, in the diagnosis of tight stenosis and occlusion. Maximal acceleration of blood flow at a tight stenosis results in Doppler signals of very high frequencies but reduced intensity. In cases of severe subclavian stenosis, in contrast to carotid stenosis, a flow signal of reduced intensity cannot be detected by CW Doppler ultrasonography because insonation of the stenosis itself is not possible. Differentiation of tight subclavian stenosis and occlusion would be possible by simultaneous ECG monitoring and latency measurements or by pulsed Doppler ultrasonography. We did not use these procedures in our routine clinical practice.

In conclusion, our data regarding the natural history of patients with subclavian steal syndrome compared with the results of surgery reveal that the indication for bypass surgery should be more restrictive, reserved for patients with disabling brainstem TIAIs since spontaneous remittance of vertebrobasilar TIAIs was approx-
imately 50%, since new TIAs took place in only 15% of initially asymptomatic persons, and since occlusion of the bypass favoring ongoing TIAs occurred in 24% of the operations.

Acknowledgments

We thank Mrs. M. Rosenberg and Mrs. R. Ehrmann for their technical support.

References

Ultrasonographic follow-up of subclavian stenosis and occlusion: natural history and surgical treatment.

H Ackermann, H C Diener, H Seboldt and C Huth

Stroke. 1988;19:431-435
doi: 10.1161/01.STR.19.4.431

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/19/4/431